Why are we so concerned with acute incomplete stent apposition?

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In their article, Shimamura et al. report that incomplete stent apposition (ISA) was observed in all 38 everolimus-eluting stents (EES) and all 39 sirolimus-eluting stents (SES) post-percutaneous coronary intervention (PCI) (measuring 315 ± 94 μm and 0.50 ± 0.24 mm² in EES and 308 ± 119 μm and 0.95 ± 0.70 mm² in SES), that it persisted in 26% of EES and 38% of SES at 8–12 months although the size of the ISA significantly decreased during follow-up in both groups (to 110 ± 165 μm and 0.17 ± 0.27 mm² in EES and 143 ± 175 μm and 0.41 ± 0.66 mm² in SES), and that the best post-stenting optical coherence tomographic (OCT)-measured ISA distance that predicted late-persistent ISA was >355 μm in EES and >285 μm in SES. The authors concluded: ‘OCT can predict late-persistent ISA after DES implantation and provide useful information to optimize PCI’.

Why are we so concerned with acute ISA? Despite the lack of supporting evidence, the most recent *Cardiac Catheterization and Interventional Cardiology Self-Assessment Program* (CathSAP) stated, ‘stent apposition may be the most important determinant of freedom from subacute stent thrombosis with DES’. To the contrary, the predictors of early ST that have been identified with intravascular imaging are primarily stent underexpansion and secondarily inflow/outflow problems such as a larger plaque burden, a small lumen area, and/or a large dissection at either stent edge. Depending on the sensitivity of the methodology used, ISA after DES implantation is observed in up to 40% by IVUS in patients undergoing primary PCI and 60–100% in stable patients by OCT. Given the nearly ubiquitous finding of acute ISA, it is not surprising that studies using IVUS or OCT have shown no relationship between acute ISA and early, late, or very late stent thrombosis after DES implantation.

Then there is the second misconception that acute ISA is important because it can persist; and late ISA has been linked to very late stent thrombosis. However, not all late ISA are equal in terms of prognosis; pathoanatomic pathways leading to late ISA also include positive remodelling causing an increase in vessel dimensions that is greater than any increase in abluminal tissue growth and abluminal thrombus dissolution or plaque regression without positive remodelling. Positive remodelling is responsible for approximately one-third of late ISA and late ISA in the setting of positive remodelling—especially large areas of malapposition (in the initial report by Cook et al., the late ISA area in their 13 very late stent thrombosis patients measured 8.3 ± 7.5 mm²) or frank aneurysm formation—is most related to stent thrombosis. While late ISA is frequently seen at the time of very late DES thrombosis, incidentally detected late ISA during routine follow-up studies of DES-treated patients is not associated with an increased frequency of subsequent adverse events. Furthermore, the cause-and-effect relationship between late ISA and very late stent thrombosis has been challenged by studies relating late ISA, very late stent thrombosis, and inflammation; studies reporting a high prevalence of strut fracture in very late stent thrombosis lesions; and most recently OCT studies indicating that neatherosclerosis may be a more important cause of very late stent thrombosis than late ISA.

What does this mean clinically? Adequately powered clinical studies—such as definitive, large registries with careful and systematic follow-up—should be undertaken to determine whether acute ISA is or is not predictive of stent thrombosis and, if so, how large an area, diameter, or volume is problematic. To the contrary, in the largest OCT study available to date in 351 patients, acute ISA was detected in 62% of stents post-PCI, measured 1.16 ± 0.69 mm² (similar to SES in the current study), persisted in half (but decreasing to 0.88 ± 0.71 mm²), and was not associated with any adverse events at 28.6 ± 10.3 months follow-up. Until information is available to the contrary, the misguided emphasis on avoiding ISA should be replaced with renewed attention to what is known to be important—stent expansion and proper lesion coverage.

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**References**

Late thrombosis of thoracic aortic stent graft

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A 17-year-old motorcycle accident victim underwent urgent successful thoracic endovascular aortic repair (TEVAR) of blunt thoracic aortic rupture. On computed tomography (CT), the rupture was distal to the origin of the subclavian artery and was treated with a custom-made, 24 mm, 6.6 cm TX2 stent. The stent graft was inserted via the femoral artery and positioned at 24 mm, 6.6 cm TX2 stent. The stent graft was treated with a custom-made, aortic repair (TEVAR) of blunt thoracic aortic rupture (Panels A, B, and see Supplementary data online, Movie S1) producing significant stenosis.

Thoracoabdominal CT showed in-stent thrombosis causing severe stenosis of the distal descending thoracic aorta, without signs of stent degeneration, endoleaks, and/or collapse of the scaffold (Panel C).

Emergency open aortic bypass surgery was performed with an extra-anatomical Dacron graft connecting the ascending aorta to the supraceliac abdominal aorta, as seen on the postoperative CT (Panel D). The patient was discharged with no additional complications.

Late thrombosis of the stent was an unusual complication of TEVAR. A possible mechanism of this complication may be a tiny intimal degeneration, endoleaks, and/or collapse of the scaffold (Panel C).

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